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                 X.25 communication option no longer available after June 2006
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                 EMBASE is now updated on a daily basis
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              AND CURRENT DISCOVER FILE IS DATED 19 DECEMBER 2005.
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=> "cartilage atrophy"

L1 33 "CARTILAGE ATROPHY"

=> 11 and pain

L2 0 L1 AND PAIN

=> 11 and osteoarthrosis

L3 0 L1 AND OSTEOARTHROSIS

=> 11 and osteoarthritis

L4 10 L1 AND OSTEOARTHRITIS

=> dup rem 14

PROCESSING COMPLETED FOR L4

L5 5 DUP REM L4 (5 DUPLICATES REMOVED)

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L5 ANSWER 1 OF 5 MEDLINE on STN DUPLICATE 1

ACCESSION NUMBER: 2003591337 MEDLINE DOCUMENT NUMBER: PubMed ID: 14673989

TITLE: Longitudinal analysis of cartilage

atrophy in the knees of patients with spinal cord

injury.

AUTHOR: Vanwanseele B; Eckstein F; Knecht H; Spaepen A; Stussi E

CORPORATE SOURCE: Swiss Federal Institute of Technology, Zurich, Switzerland.. vanwanseele@biomech.mat.ethz.ch

SOURCE: Arthritis and rheumatism, (2003 Dec) Vol. 48, No. 12, pp.

3377-81.

Journal code: 0370605. ISSN: 0004-3591.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 200401

ENTRY DATE: Entered STN: 20031216

Last Updated on STN: 20040115 Entered Medline: 20040114

AB OBJECTIVE: A previous cross-sectional study indicated that the morphology of patellar and tibial cartilage is subject to change after spinal cord injury (SCI). The aim of this study was to perform a longitudinal analysis of cartilage atrophy in all knee compartments, including the femoral condyles, in SCI patients over 12 months. METHODS: The right knees of 9 patients with complete, traumatic SCI were examined shortly after the injury (mean +/- SD 9 +/- 4 weeks) and at 6 and 12 months postinjury. Three-dimensional morphology of the patellar, tibial, and femoral cartilage (mean and maximum thickness,

volume, and surface area) was determined from coronal and transversal

magnetic resonance images (fat-suppressed gradient-echo sequences) using validated postprocessing techniques. RESULTS: The mean thickness of knee joint cartilage decreased significantly during the first 6 months after injury (range 5-7%; P < 0.05). The mean change at 12 months was 9% in the patella, 11% in the medial tibia, 11% in the medial femoral condyle, 13% in the lateral tibia, and 10% in the lateral femoral condyle (P < 0.05 for all compartments). CONCLUSION: This is the first report of a longitudinal analysis of cartilage atrophy in patients with SCI. These data show that human cartilage atrophies in the absence of normal joint loading and movement after SCI, with a rate of change that is higher than that observed in osteoarthritis (OA). A potential clinical implication is that cartilage thinning after SCI may affect the stress distribution in the joint and render it vulnerable to OA. Future studies should focus on whether specific exercise protocols and rehabilitation programs can prevent cartilage thinning.

L5 ANSWER 2 OF 5 MEDLINE on STN . DUPLICATE 2

ACCESSION NUMBER: 92208668 MEDLINE DOCUMENT NUMBER: PubMed ID: 1555051

TITLE: A mini review: proteoglycan aggregate profiles in the

Pond-Nuki dog model of osteoarthritis and in

canine disuse atrophy.

AUTHOR: Howell D S; Muller F; Manicourt D H

CORPORATE SOURCE: Department of Medicine, University of Miami School of

Medicine, FL 33101.

SOURCE: British journal of rheumatology, (1992) Vol. 31 Suppl 1,

pp. 7-11. Ref: 24

Journal code: 8302415. ISSN: 0263-7103.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 199205

ENTRY DATE: Entered STN: 19920515

Last Updated on STN: 19920515 Entered Medline: 19920507

ΑB The Pond-Nuki dog model of osteoarthritis has characteristics which seem to mimic the human disease in early stages, particularly with respect to progressive changes in the cartilage matrix. Aggregating proteoglycans were studied using novel extraction and ultracentrifugation methods designed to separate very large macromolecules. With these methods two large peaks of proteoglycan (PG) aggregates (PGA-1 and PGA-2) were separated in preparative amounts and were shown to have unequivocal differences in composition in many respects. The profiles of these peaks have been studied as a function of joint location, topographic site, cartilage layer, presence of cartilage atrophy versus osteoarthritis, as well as treatment of the animals with various agents. Both link protein (essential for forming link-protein stabilized aggregates) and hyaluronate are required to regenerate normal aggregate profiles from the deficient aggregate fractions obtained from osteoarthritic cartilage. Canine proteoglycan link-stabilized aggregates (PGA-2) are confined to the middle and deep zone of cartilage. We believe that their reduction or elimination in the Pond-Nuki model results from a disturbance or loss of functional link protein (and hyaluronate), thereby weakening the middle and deep cartilage layers.

L5 ANSWER 3 OF 5 MEDLINE on STN DUPLICATE 3

ACCESSION NUMBER: 90037565 MEDLINE DOCUMENT NUMBER: PubMed ID: 2808699

TITLE: Cartilage matrix glycoprotein is present in serum in

experimental canine osteoarthritis.

AUTHOR: Fife R S; Brandt K D

CORPORATE SOURCE: Department of Medicine, Indiana University School of

Medicine, Indianapolis 46202.

CONTRACT NUMBER: AR-20582 (NIAMS)

AR-34367 (NIAMS) AR-39250 (NIAMS)

SOURCE: The Journal of clinical investigation, (1989 Nov) Vol. 84,

No. 5, pp. 1432-9.

Journal code: 7802877. ISSN: 0021-9738.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 198912

ENTRY DATE: Entered STN: 19900328

> Last Updated on STN: 19970203 Entered Medline: 19891204

AB We have described previously a disulfide-bonded 550,000-D cartilage matrix glycoprotein (CMGP), which is found in normal hyaline cartilage, fibrocartilage, and the vitreous of the eye, and consists of subunits with apparent molecular weights of 130,000 in 4% gels (116,000 in 9% gels). In osteoarthritic cartilage from dogs subjected to transection of the anterior cruciate ligament (ACL), CMGP is cleaved to major immunoreactive fragments with apparent molecular weights of 65,000 and 75,000 after reduction with 2-mercaptoethanol. In the present study, using immunolocation analysis, a monoclonal antibody to CMGP did not react with serum from 8 of 12 dogs before ACL transection but did react with serum from seven of these animals 4 wk after surgery and with serum from 10 dogs at sacrifice, 8-14 wk after ACL transection. Serum from four dogs reacted with the monoclonal antibody before ACL transection. Serum from two dogs was negative at all time points. Immunolocation studies using a polyclonal antiserum to CMGP were performed in seven of these dogs and produced results identical with the monoclonal antibody in four dogs. In contrast, analysis of serial serum samples from three dogs with cartilage atrophy revealed no evidence of CMGP at any time point. These data suggest that CMGP may be a serum marker for osteoarthritis in this canine model.

L5 ANSWER 4 OF 5 MEDLINE on STN DUPLICATE 4

ACCESSION NUMBER: 85045915

MEDLINE

DOCUMENT NUMBER: PubMed ID: 6497604 TITLE:

Synovectomy as treatment for purulent joint infection. AUTHOR:

Tscherne H; Giebel G; Muhr G; Howell C

SOURCE: Archives of orthopaedic and traumatic surgery. Archiv fur orthopadische und Unfall-Chirurgie, (1984) Vol. 103, No. 3,

pp. 162-4.

Journal code: 7803037. ISSN: 0344-8444. GERMANY, WEST: Germany, Federal Republic of

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

PUB. COUNTRY:

FILE SEGMENT: Priority Journals

ENTRY MONTH: 198412

ENTRY DATE: Entered STN: 19900320

Last Updated on STN: 19900320 Entered Medline: 19841220

AB Conventional treatment of pyogenic knee joint infections leads to unsatisfactory results. Through early synovectomy, before cartilage damage and osteoarthritis appear, the infected focus can be "excised." Functional after-treatment avoids cartilage atrophy, wound adhesions, and muscle weakness. The excellent results after 26 knee joint infections confirm this.

ANSWER 5 OF 5 MEDLINE on STN DUPLICATE 5

ACCESSION NUMBER: 84277727 MEDLINE DOCUMENT NUMBER: PubMed ID: 6465163

TITLE: Effects of salicylates and other nonsteroidal

anti-inflammatory drugs on articular cartilage.

AUTHOR: Brandt K D; Palmoski M J CONTRACT NUMBER: AM 20582 (NIADDK)

AM 27075 (NIADDK)

SOURCE: The American journal of medicine, (1984 Jul 13) Vol. 77,

No. 1A, pp. 65-9.

Journal code: 0267200. ISSN: 0002-9343.

PUB. COUNTRY:

United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH:

198409

ENTRY DATE:

Entered STN: 19900320

Last Updated on STN: 19970203 Entered Medline: 19840907

AB According to in vivo experimental data, salicylates and several other nonsteroidal anti-inflammatory agents suppress proteoglycan biosynthesis in normal and degenerating articular cartilage. Therapeutic levels of aspirin in vivo had a similar adverse effect on degenerating cartilage, as noted in two canine models of osteoarthritis and cartilage atrophy. Because the effective daily antirheumatic dose of nonsteroidal anti-inflammatory drugs is lower than that of salicylates, these drugs may have less negative effects on degenerating articular cartilage. However, clinical significance cannot be extrapolated from these experimental data.

=> d ibib abs total 11

L1 ANSWER 1 OF 33 MEDLINE on STN

ACCESSION NUMBER: 2
DOCUMENT NUMBER: P

2004427822 MEDLINE PubMed ID: 15334465

TITLE:

Cartilage atrophy in the knees of

patients after seven weeks of partial load bearing.

AUTHOR:

Hinterwimmer S; Krammer M; Krotz M; Glaser C; Baumgart R;

Reiser M; Eckstein F

CORPORATE SOURCE:

SOURCE:

Ludwig-Maximilians-Universitat Munchen, Munich, Germany. Arthritis and rheumatism, (2004 Aug) Vol. 50, No. 8, pp.

2516-20.

Journal code: 0370605. ISSN: 0004-3591.

PUB. COUNTRY:

United States

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH:

200409

ENTRY DATE:

Entered STN: 20040831

Last Updated on STN: 20040925 Entered Medline: 20040924

AB OBJECTIVE: It is currently unknown whether human cartilage properties change during short periods of partial load bearing. We used a post-ankle fracture model to explore whether changes in cartilage morphology occur in the knee under conditions of partial load bearing. METHODS: The knees of 20 patients with Weber type B and type C fractures were examined using magnetic resonance imaging. The first scan was obtained shortly (mean +/-SD 3.2 +/- 3.0 days) after the injury, and a second scan was obtained 7weeks later (mean \pm +/- SD 50.7 \pm /- 5.5 days). The morphology (mean and maximum thickness, volume, and surface area) of the patellar, tibial, and femoral cartilage was determined from coronal and axial magnetic resonance images (fat-suppressed gradient-echo). RESULTS: Between week 0 and week 7, the cross-sectional area of the quadriceps muscle was reduced by 11% (P<0.001). Changes in the mean (+/-SD) cartilage thickness ranged from -2.9 +/- 3.2% in the patella to -6.6 +/- 4.9% in the medial tibia. No significant change in cartilage morphology of the contralateral knee was observed. CONCLUSION: Results of this study demonstrate that in a post-ankle fracture model of partial load bearing, cartilage morphology in all knee compartments is subject to significant change. Changes in the femorotibial joint exceeded those in the patella, whereas no change was

observed in the contralateral knee. These findings raise the question of whether cartilage is mechanically less competent and particularly vulnerable after states of partial or complete immobilization.

L1 ANSWER 2 OF 33 MEDLINE on STN ACCESSION NUMBER: 2003591337 MEDLINE DOCUMENT NUMBER: PubMed ID: 14673989

TITLE: Longitudinal analysis of cartilage

atrophy in the knees of patients with spinal cord

injury.

AUTHOR: Vanwanseele B; Eckstein F; Knecht H; Spaepen A; Stussi E

CORPORATE SOURCE: Swiss Federal Institute of Technology, Zurich,

Switzerland.. vanwanseele@biomech.mat.ethz.ch

SOURCE: Arthritis and rheumatism, (2003 Dec) Vol. 48, No. 12, pp.

3377-81.

Journal code: 0370605. ISSN: 0004-3591.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 200401

ENTRY DATE: Entered STN: 20031216

Last Updated on STN: 20040115 Entered Medline: 20040114

AB OBJECTIVE: A previous cross-sectional study indicated that the morphology of patellar and tibial cartilage is subject to change after spinal cord injury (SCI). The aim of this study was to perform a longitudinal analysis of cartilage atrophy in all knee compartments, including the femoral condyles, in SCI patients over 12 months. METHODS: The right knees of 9 patients with complete, traumatic SCI were examined shortly after the injury (mean +/- SD 9 +/- 4 weeks) and at 6 and 12 months postinjury. Three-dimensional morphology of the patellar, tibial, and femoral cartilage (mean and maximum thickness, volume, and surface area) was determined from coronal and transversal magnetic resonance images (fat-suppressed gradient-echo sequences) using validated postprocessing techniques. RESULTS: The mean thickness of knee joint cartilage decreased significantly during the first 6 months after injury (range 5-7%; P < 0.05). The mean change at 12 months was 9% in the patella, 11% in the medial tibia, 11% in the medial femoral condyle, 13% in the lateral tibia, and 10% in the lateral femoral condyle (P < 0.05 for all compartments). CONCLUSION: This is the first report of a longitudinal analysis of cartilage atrophy in patients with SCI. These data show that human cartilage atrophies in the absence of normal joint loading and movement after SCI, with a rate of change that is higher than that observed in osteoarthritis (OA). A potential clinical implication is that cartilage thinning after SCI may affect the stress distribution in the joint and render it vulnerable to Future studies should focus on whether specific exercise protocols and rehabilitation programs can prevent cartilage thinning.

L1 ANSWER 3 OF 33 MEDLINE ON STN ACCESSION NUMBER: 2003050918 MEDLINE DOCUMENT NUMBER: PubMed ID: 12560716

TITLE: Reinforced orbitotemporal lift: contribution to midface

rejuvenation.

AUTHOR: Reno Waldir Teixeira

CORPORATE SOURCE: Plastic Surgery Service at Santa Casa, Misericordia de

Guaratingueta Hospital, Sao Paulo, Brazil.

SOURCE: Plastic and reconstructive surgery, (2003 Feb) Vol. 111,

No. 2, pp. 869-77; discussion 878-9.

Journal code: 1306050. ISSN: 0032-1052.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH:

200302

ENTRY DATE:

Entered STN: 20030202

Last Updated on STN: 20030227 Entered Medline: 20030226

AB The changes in the aging face occur from progressive ptosis of the skin, fat, and muscle, in conjunction with bone absorption and cartilage In the orbital region, hollowness and compartmentalization occur. Conventional face lift procedures correct only the skin flaccidity, and superficial musculoaponeurotic system techniques reposition the skin and platysma without repositioning the middle third of the face, creating an artificial jawline. Subperiosteal . rhytidectomy disrupts the anatomy of the periorbita, which gives the patient a certain scarecrow aspect. Composite rhytidectomy associated with brow lift and blepharoplasty may offer better results, with improvement in the malar and orbital regions. The reinforced orbitotemporal lift (ROTEL) is a new procedure in a face lift that allows the orbicularis oculi muscle and all the structures connected to it to be elevated and stretched and the orbitotemporal skin to be raised, repositioning these structures and ending orbital compartmentalization. The result is an impressive improvement in the malar-orbitotemporal

L1ANSWER 4 OF 33

MEDLINE on STN

region, resulting in a natural and youthful appearance.

ACCESSION NUMBER:

2000204118 MEDLINE

DOCUMENT NUMBER:

PubMed ID: 10738181

TITLE:

Bronchial cartilage atrophy in chronic

bronchitis: observations on chondrolytic processes.

AUTHOR:

Tetlow L C; Freemont A J; Woolley D E

CORPORATE SOURCE:

University Department of Medicine, Manchester Royal Infirmary, Manchester, UK.. lynne.c.tetlow@man.ac.uk

SOURCE:

Pathobiology: journal of immunopathology, molecular and

cellular biology, (1999 Jul-Aug) Vol. 67, No. 4, pp.

196-201.

Journal code: 9007504. ISSN: 1015-2008.

PUB. COUNTRY:

Switzerland

DOCUMENT TYPE:

Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

200004

ENTRY DATE:

Entered STN: 20000505

Last Updated on STN: 20000505 Entered Medline: 20000426

AB The status of bronchial cartilage degeneration in chronic bronchitis is unclear, and little is known about the chondrolytic mechanisms involved. The potential contributions of various inflammatory cells, chondrocytes and cartilage-degrading enzymes to cartilage atrophy have been examined. Bronchial cartilage specimens were obtained at autopsy from lobar secondary bronchi from chronic bronchitics and age-matched controls; each was examined by light microscopy and immunohistology for the distributions of mast cells, macrophages, eosinophils, collagenase 1, collagenase 3, and degradation products of cartilage collagen. Most bronchitic specimens showed hypertrophic chondrocytes, some of which were immunostained for collagenase 3, and occasionally for collagenase 1. Evidence for collagen degradation products was demonstrated around the lacunae of a proportion of chondrocytes, and both collagenases were also observed in the soft inflammatory tissues in close association with the cartilage surface, together with variable distributions of mast cells and macrophages. observations were generally absent or very much reduced in the control, non-bronchitic specimens. Degenerative changes, atrophy and loss of bronchial cartilage were common features of most chronic bronchitic specimens, this usually being related to intrinsic changes in the chondrocyte phenotype, including proliferative and matrix-degrading properties. Mast cells and macrophages were often observed in close association with the bronchial cartilage, suggesting that inflammatory

cells may also contribute to the mechanisms of bronchial cartilage degradation and loss. These observations of bronchial cartilage degeneration were generally lacking in age-matched non-bronchitic control specimens.

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L1 ANSWER 5 OF 33 MEDLINE on STN ACCESSION NUMBER: 1999267782 MEDLINE DOCUMENT NUMBER: PubMed ID: 10335301

TITLE: Remobilization does not fully restore immobilization

induced articular cartilage atrophy.

AUTHOR: Haapala J; Arokoski J P; Hyttinen M M; Lammi M; Tammi M;

Kovanen V; Helminen H J; Kiviranta I

CORPORATE SOURCE: Department of Surgery, Kuopio University Hospital, Finland. SOURCE: Clinical orthopaedics and related research, (1999 May) No.

362, pp. 218-29.

Journal code: 0075674. ISSN: 0009-921X.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 199906

ENTRY DATE: Entered STN: 19990618

Last Updated on STN: 19990618 Entered Medline: 19990608

AB The recovery of articular cartilage from immobilization induced atrophy was studied. The right hind limbs of 29-week-old beagle dogs were immobilized for 11 weeks and then remobilized for 50 weeks. Cartilage from the immobilized knee was compared with tissue from age matched control animals. After the immobilization period, uncalcified articular cartilage glycosaminoglycan concentration was reduced by 20% to 23%, the reduction being largest (44%) in the superficial zone. The collagen fibril network showed no significant changes, but the amount of collagen crosslinks was reduced (13.5%) during immobilization. After remobilization, glycosaminoglycan concentration was restored at most sites, except for in the upper parts of uncalcified cartilage in the medial femoral and tibial condyles (9% to 17% less glycosaminoglycans than in controls). The incorporation of 35SO4 was not changed, and remobilization also did not alter the birefringence of collagen fibrils. Remobilization restored the proportion of collagen crosslinks to the control level. The changes induced by joint unloading were reversible at most sites investigated, but full restoration of articular cartilage glycosaminoglycan concentration was not obtained in all sites, even after remobilization for 50 weeks. This suggests that lengthy immobilization of a joint can cause long lasting articular cartilage proteoglycan alterations at the same time as collagen organization remains largely unchanged. Because proteoglycans exert strong influence on the biomechanical properties of cartilage, lengthy immobilization may jeopardize the well being of articular cartilage.

L1 ANSWER 6 OF 33 MEDLINE on STN ACCESSION NUMBER: 92208668 MEDLINE DOCUMENT NUMBER: PubMed ID: 1555051

TITLE: A mini review: proteoglycan aggregate profiles in the

Pond-Nuki dog model of osteoarthritis and in canine disuse

atrophy.

AUTHOR: Howell D S; Muller F; Manicourt D H

CORPORATE SOURCE: Department of Medicine, University of Miami School of

Medicine, FL 33101.

SOURCE: British journal of rheumatology, (1992) Vol. 31 Suppl 1,

pp. 7-11. Ref: 24

Journal code: 8302415. ISSN: 0263-7103.

PUB. COUNTRY: ENGLAND: United Kingdom

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 199205

ENTRY DATE: Entered STN: 19920515

Last Updated on STN: 19920515 Entered Medline: 19920507

AB The Pond-Nuki dog model of osteoarthritis has characteristics which seem to mimic the human disease in early stages, particularly with respect to progressive changes in the cartilage matrix. Aggregating proteoglycans were studied using novel extraction and ultracentrifugation methods designed to separate very large macromolecules. With these methods two large peaks of proteoglycan (PG) aggregates (PGA-1 and PGA-2) were separated in preparative amounts and were shown to have unequivocal differences in composition in many respects. The profiles of these peaks have been studied as a function of joint location, topographic site, cartilage layer, presence of cartilage atrophy versus osteoarthritis, as well as treatment of the animals with various agents. Both link protein (essential for forming link-protein stabilized aggregates) and hyaluronate are required to regenerate normal aggregate profiles from the deficient aggregate fractions obtained from osteoarthritic cartilage. Canine proteoglycan link-stabilized aggregates (PGA-2) are confined to the middle and deep zone of cartilage. We believe that their reduction or elimination in the Pond-Nuki model results from a disturbance or loss of functional link protein (and hyaluronate), thereby weakening the middle and deep cartilage layers.

L1 ANSWER 7 OF 33 MEDLINE on STN ACCESSION NUMBER: 90382043 MEDLINE DOCUMENT NUMBER: PubMed ID: 2205438

TITLE: Pathophysiology of chronic obstructive pulmonary disease.

AUTHOR: Thurlbeck W M

CORPORATE SOURCE: Department of Pathology, University of British Columbia,

Vancouver, Canada.

SOURCE: Clinics in chest medicine, (1990 Sep) Vol. 11, No. 3, pp.

389-403. Ref: 78

Journal code: 7907612. ISSN: 0272-5231.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 199010

ENTRY DATE: Entered STN: 19901122

Last Updated on STN: 19970203 Entered Medline: 19901026

AB Chronic airflow obstruction (CAO) is a syndrome that is produced by a variety of lesions which may occur in bronchi (large airways), bronchioles (small airways), and lung parenchyma (gas exchanging lung). These lesions frequently occur together in various combinations because of a common etiologic agent, tobacco smoke. Occasionally, one lesion or another may play a dominant role. The major disease of the large airways is chronic bronchitis, or chronic sputum production, and it is defined clinically. Its morphologic counterpart is mucous gland enlargement. Mucous gland enlargement is poorly related to CAO. Other lesions of the large airways--inflammation, smooth muscle hyperplasia, cartilage atrophy, and bronchial wall thickening--have also been described, but their functional consequences are uncertain. Bronchiolar lesions are well recognized in CAO, but their relative importance may differ in patients with mild CAO, compared to patients with severe CAO. CAO, inflammation is a very important lesion, and its probable consequences--narrowing, fibrosis, and goblet cell metaplasia--have all been found to be important. In severe CAO, inflammation and fibrosis do not appear to be important, but goblet cell metaplasia, bronchiolar tortuosity, and narrowing do. Emphysema is a subset of airspace enlargement. Emphysema is defined anatomically and is the most important

component of severe CAO. Several forms of emphysema can be recognized morphologically and may have specific clinical associations. However, in the usual patient with severe CAO, it is the severity, rather than the type, of emphysema, that is most significant. The diagnosis of emphysema depends on a combined approach. Significant factors include the clinical history (age, sex, smoking, chronic bronchitis, dyspnea), radiologic evidence of overinflation, and diminished diffusing capacity for carbon monoxide.

L1 ANSWER 8 OF 33 MEDLINE on STN ACCESSION NUMBER: 90125994 MEDLINE DOCUMENT NUMBER: PubMed ID: 2404712

TITLE: Pathology of chronic airflow obstruction.

AUTHOR: Thurlbeck W M

CORPORATE SOURCE: University of British Columbia, Faculty of Medicine,

Vancouver, Canada.

SOURCE: Chest, (1990 Feb) Vol. 97, No. 2 Suppl, pp. 6S-10S. Ref:

11

Journal code: 0231335. ISSN: 0012-3692.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 199003

ENTRY DATE: Entered STN: 19900328

Last Updated on STN: 19900328 Entered Medline: 19900313

AB Classification of chronic airflow obstruction may be based on the site of the obstructing lesions. It is seldom that only one type of lesion is present, but one may often dominate. In chronic bronchitis, the major disease of large airways, chronic mucus hypersecretion, is reflected by an increase in size of bronchial mucous glands. This may be a factor in airway narrowing, especially with coexisting edema of the airway wall. Excess intralumenal mucus compounds the obstruction. Increased airways reactivity is present in 15 to 70 percent of patients with chronic airflow obstruction. Increased airway muscle and cartilage atrophy are features of chronic bronchitis, but the association of increased muscle with increased airway reactivity is poor. Inflammation of the small airways (bronchiolitis) is a significant complication for cigarette smokers and is an important cause of mild chronic airflow obstruction. Goblet cell metaplasia is a reflection of chronic small airways inflammation and, together with intralumenal mucus, is an important feature. Permanent narrowing of the small airways presumably results from inflammation with consequent fibrosis, while functional narrowing results from release of mediators of inflammation. Increased muscle mass is present in some cases. Distortion and irregularity of small airways related to emphysema are major factors in severe obstruction. Lesser degrees of emphysema may be associated with a diminished number of alveolar attachments and mild chronic airflow obstruction. Emphysema, the dominant lesion in patients with severe chronic airflow obstruction, results from parenchymal lesions. Centrilobular emphysema, in which the respiratory bronchioles are selectively or dominantly involved, is the most common form. (ABSTRACT TRUNCATED AT 250 WORDS)

L1 ANSWER 9 OF 33 MEDLINE on STN ACCESSION NUMBER: 90037565 MEDLINE DOCUMENT NUMBER: PubMed ID: 2808699

TITLE: Cartilage matrix glycoprotein is present in serum in

experimental canine osteoarthritis.

AUTHOR: Fife R S; Brandt K D

CORPORATE SOURCE: Department of Medicine, Indiana University School of

Medicine, Indianapolis 46202.

CONTRACT NUMBER: AR-20582 (NIAMS)

AR-34367 (NIAMS) AR-39250 (NIAMS)

SOURCE: The Journal of clinical investigation, (1989 Nov) Vol. 84,

No. 5, pp. 1432-9.

Journal code: 7802877. ISSN: 0021-9738.

PUB. COUNTRY:

United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE:

English

FILE SEGMENT:

ENTRY MONTH:

Abridged Index Medicus Journals; Priority Journals 198912

ENTRY DATE:

Entered STN: 19900328

Last Updated on STN: 19970203 Entered Medline: 19891204

AB We have described previously a disulfide-bonded 550,000-D cartilage matrix glycoprotein (CMGP), which is found in normal hyaline cartilage, fibrocartilage, and the vitreous of the eye, and consists of subunits with apparent molecular weights of 130,000 in 4% gels (116,000 in 9% gels). In osteoarthritic cartilage from dogs subjected to transection of the anterior cruciate ligament (ACL), CMGP is cleaved to major immunoreactive fragments with apparent molecular weights of 65,000 and 75,000 after reduction with 2-mercaptoethanol. In the present study, using immunolocation analysis, a monoclonal antibody to CMGP did not react with serum from 8 of 12 dogs before ACL transection but did react with serum from seven of these animals $4\ \mathrm{wk}$ after surgery and with serum from $10\ \mathrm{dogs}$ at sacrifice, 8-14 wk after ACL transection. Serum from four dogs reacted with the monoclonal antibody before ACL transection. Serum from two dogs was negative at all time points. Immunolocation studies using a polyclonal antiserum to CMGP were performed in seven of these dogs and produced results identical with the monoclonal antibody in four dogs. In contrast, analysis of serial serum samples from three dogs with cartilage atrophy revealed no evidence of CMGP at any

time point. These data suggest that CMGP may be a serum marker for osteoarthritis in this canine model.

L1 ANSWER 10 OF 33 MEDLINE on STN ACCESSION NUMBER: 85045915 MEDLINE DOCUMENT NUMBER: PubMed ID: 6497604

TITLE:

Synovectomy as treatment for purulent joint infection.

AUTHOR:

Tscherne H; Giebel G; Muhr G; Howell C

SOURCE:

Archives of orthopaedic and traumatic surgery. Archiv fur

orthopadische und Unfall-Chirurgie, (1984) Vol. 103, No. 3,

pp. 162-4.

Journal code: 7803037. ISSN: 0344-8444. GERMANY, WEST: Germany, Federal Republic of Journal; Article; (JOURNAL ARTICLE)

PUB. COUNTRY: DOCUMENT TYPE:

LANGUAGE:

English

FILE SEGMENT:

Priority Journals

ENTRY MONTH:

198412

ENTRY DATE:

Entered STN: 19900320

Last Updated on STN: 19900320 Entered Medline: 19841220

AB Conventional treatment of pyogenic knee joint infections leads to unsatisfactory results. Through early synovectomy, before cartilage damage and osteoarthritis appear, the infected focus can be "excised." Functional after-treatment avoids cartilage atrophy, wound adhesions, and muscle weakness. The excellent results after 26 knee joint infections confirm this.

ANSWER 11 OF 33 MEDLINE on STN ACCESSION NUMBER: 84277727 MEDLINE DOCUMENT NUMBER: PubMed ID: 6465163

TITLE: Effects of salicylates and other nonsteroidal

anti-inflammatory drugs on articular cartilage.

AUTHOR:

Brandt K D; Palmoski M J

CONTRACT NUMBER:

AM 20582 (NIADDK)

AM 27075 (NIADDK)

SOURCE: The American journal of medicine, (1984 Jul 13) Vol. 77,

No. 1A, pp. 65-9.

Journal code: 0267200. ISSN: 0002-9343.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 198409

ENTRY DATE: Entered STN: 19900320

Last Updated on STN: 19970203 Entered Medline: 19840907

AB According to in vivo experimental data, salicylates and several other nonsteroidal anti-inflammatory agents suppress proteoglycan biosynthesis in normal and degenerating articular cartilage. Therapeutic levels of aspirin in vivo had a similar adverse effect on degenerating cartilage, as noted in two canine models of osteoarthritis and cartilage atrophy. Because the effective daily antirheumatic dose of nonsteroidal anti-inflammatory drugs is lower than that of salicylates, these drugs may have less negative effects on degenerating articular cartilage. However, clinical significance cannot be extrapolated from these experimental data.

L1 ANSWER 12 OF 33 MEDLINE ON STN ACCESSION NUMBER: 82160255 MEDLINE DOCUMENT NUMBER: PubMed ID: 7066039

TITLE: Articular cartilage atrophy in lower

limb amputees.

AUTHOR: Benichou C; Wirotius J M

SOURCE: Arthritis and rheumatism, (1982 Jan) Vol. 25, No. 1, pp.

80-2.

Journal code: 0370605. ISSN: 0004-3591.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 1982,05

ENTRY DATE: Entered STN: 19900317

Last Updated on STN: 19900317 Entered Medline: 19820512

AB A retrospective and radiologic survey of the hips of 53 above-knee amputees showed that none of these hips was normal. Osteoporosis was present in all subjects, and cartilage thickness was reduced in 27 cases. This reduced thickness was inversely correlated with stump length, since it occurred in 11 of 13 upper-third amputees, but in none of 10 lower-third amputees. The mechanisms of cartilage atrophy are discussed.

L1 ANSWER 13 OF 33 MEDLINE ON STN ACCESSION NUMBER: 82091306 MEDLINE DOCUMENT NUMBER: PubMed ID: 7317111

TITLE: Running inhibits the reversal of atrophic changes in canine

knee cartilage after removal of a leg cast.

AUTHOR: Palmoski M J; Brandt K D

CONTRACT NUMBER: AM 20582 (NIADDK)

RACT NUMBER: AM 20582 (NIADDK)
AM 27075 (NIADDK)

SOURCE: Arthritis and rheumatism, (1981 Nov) Vol. 24, No. 11, pp.

1329-37.

Journal code: 0370605. ISSN: 0004-3591.

Report No.: NASA-82091306.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals; Space

Life Sciences

ENTRY MONTH: 198202

ENTRY DATE: Entered STN: 19900316

Last Updated on STN: 19970203 Entered Medline: 19820222

AΒ The effect of vigorous exercise on the reversibility of canine knee cartilage atrophy produced by immobilization of the leg was studied. In comparison to cartilage from the contralateral control knees, cartilage from knees which had been immobilized in a cast for 6 weeks showed an increase in water content and decreases in thickness, Safranin O staining of the matrix, uronic acid content, and net proteoglycan synthesis. In addition, the ability of both newly synthesized (35S) and total tissue proteoglycans to interact with hyaluronic acid to form aggregates was diminished; this was apparently due to an abnormality in the hyaluronate-binding region of the core proteins. If the casts were removed and the animals were then allowed to ambulate ad libitum for 3 weeks, all of these changes were reversed. However, knee cartilage from 3 dogs which had been run daily on a treadmill (6 miles/day) for 3 weeks after removal of the casts exhibited continuing decreases in thickness, Safranin O staining, and uronic acid content (mean 31%), even though net proteoglycan synthesis was increased (mean 16%) in comparison to that in control cartilage from the contralateral (nonimmobilized) knee. Furthermore, the abnormality in both 35S- and total tissue proteoglycans which precluded their interaction with high molecular weight hyaluronic acid persisted. In this respect, the proteoglycans were indistinguishable from those obtained from knee cartilage immediately following 6 weeks in a cast.

L1 ANSWER 14 OF 33 MEDLINE on STN ACCESSION NUMBER: 81281523 MEDLINE DOCUMENT NUMBER: PubMed ID: 7271631

TITLE: Cartilage atrophy following spinal cord

damage.

AUTHOR: Anderson J; Breidahl P

SOURCE: Australasian radiology, (1981 Mar) Vol. 25, No. 1, pp.

98-103.

Journal code: 0047441. ISSN: 0004-8461.

PUB. COUNTRY: Australia

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Priority Journals

ENTRY MONTH: 198110

ENTRY DATE: Entered STN: 19900316

Last Updated on STN: 19900316 Entered Medline: 19811014

L1 ANSWER 15 OF 33 MEDLINE ON STN ACCESSION NUMBER: 74177067 MEDLINE DOCUMENT NUMBER: PubMed ID: 4832511 TITLE: Cartilage atrophy.

AUTHOR: Pool W H Jr

SOURCE: Radiology, (1974 Jul) Vol. 112, No. 1, pp. 47-50.

Journal code: 0401260. ISSN: 0033-8419.

PUB. COUNTRY: United States

DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English

FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals

ENTRY MONTH: 197407

ENTRY DATE: Entered STN: 19900310

Last Updated on STN: 19900310 Entered Medline: 19740730

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ACCESSION NUMBER: 2004344653 EMBASE

TITLE: Cartilage atrophy in the knees of

patients after seven weeks of partial load bearing.

Hinterwimmer S.; Krammer M.; Krotz M.; Glaser C.; Baumgart R.; Reiser M.; Eckstein F.

Dr. F. Eckstein, Institute of Anatomy, Paracelsus Priv. Medical University, Strubergasse A2, A5020 Salzburg,

Germany. Felix.Eckstein@pmu.ac.at

SOURCE: Arthritis and Rheumatism, (2004) Vol. 50, No. 8, pp.

2516-2520. . Refs: 16

ISSN: 0004-3591 CODEN: ARHEAW

COUNTRY: United States Journal; Article DOCUMENT TYPE: FILE SEGMENT:

014 Radiology

031 Arthritis and Rheumatism

LANGUAGE: English SUMMARY LANGUAGE: English

AUTHOR:

CORPORATE SOURCE:

ENTRY DATE: Entered STN: 9 Sep 2004

Last Updated on STN: 9 Sep 2004

AΒ Objective. It is currently unknown whether human cartilage properties change during short periods of partial load bearing. We used a post-ankle fracture model to explore whether changes in cartilage morphology occur in the knee under conditions of partial load bearing. Methods. The knees of 20 patients with Weber type B and type C fractures were examined using magnetic resonance imaging. The first scan was obtained shortly (mean \pm SD 3.2 \pm 3.0 days) after the injury, and a second scan was obtained 7 weeks later (mean ± SD 50.7 ± 5.5 days). The morphology (mean and maximum thickness, volume, and surface area) of the patellar, tibial, and femoral cartilage was determined from coronal and axial magnetic resonance images (fat-suppressed gradient-echo). Results. Between week 0 and week 7, the cross-sectional area of the quadriceps muscle was reduced by 11% (P<0.001). Changes in the mean $(\pm SD)$ cartilage thickness ranged from $-2.9 \pm 3.2\%$ in the patella to $-6.6 \pm$ 4.9% in the medial tibia. No significant change in cartilage morphology of the contralateral knee was observed. Conclusion. Results of this study demonstrate that in a post-ankle fracture model of partial load bearing, cartilage morphology in all knee compartments is subject to significant change. Changes in the femorotibial joint exceeded those in the patella, whereas no change was observed in the contralateral knee. These findings raise the question of whether cartilage is mechanically less competent and particularly vulnerable after states of partial or complete immobilization.

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ACCESSION NUMBER: 2003511075 EMBASE

TITLE: Longitudinal Analysis of Cartilage

Atrophy in the Knees of Patients with Spinal Cord

Injury.

Vanwanseele B.; Eckstein F.; Knecht H.; Spaepen A.; Stussis AUTHOR:

> B. Vanwanseele, Laboratory for Biomechanics, ETHZ, Wagistrasse 4, Schlieren CH-8652, Switzerland.

vanwanseele@biomech.mat.ethz.ch

SOURCE: Arthritis and Rheumatism, (2003) Vol. 48, No. 12, pp.

> 3377-3381. . Refs: 15

ISSN: 0004-3591 CODEN: ARHEAW

COUNTRY: United States DOCUMENT TYPE: Journal; Article

FILE SEGMENT: Arthritis and Rheumatism 031

> Orthopedic Surgery 033

LANGUAGE: English SUMMARY LANGUAGE: English

CORPORATE SOURCE:

ENTRY DATE: Entered STN: 16 Jan 2004

Last Updated on STN: 16 Jan 2004

Objective. A previous cross-sectional study indicated that the morphology AB of patellar and tibial cartilage is subject to change after spinal cord injury (SCI). The aim of this study was to perform a longitudinal analysis of cartilage atrophy in all knee compartments, including the femoral condyles, in SCI patients over 12 months. Methods. The right knees of 9 patients with complete, traumatic SCI were examined shortly after the injury (mean ± SD 9 ± 4 weeks) and at 6 and 12 months postinjury. Three-dimensional morphology of the patellar, tibial, and femoral cartilage (mean and maximum thickness, volume, and surface area) was determined from coronal and transversal magnetic resonance images (fat-suppressed gradient-echo sequences) using validated postprocessing techniques. Results. The mean thickness of knee joint cartilage decreased significantly during the first 6 months after injury (range 5-7%; P < 0.05). The mean change at 12 months was 9% in the patella, 11% in the medial tibia, 11% in the medial femoral condyle, 13% in the lateral tibia, and 10% in the lateral femoral condyle (P < 0.05 for all compartments). Conclusion. This is the first report of a longitudinal analysis of cartilage atrophy in patients with SCI. These data show that human cartilage atrophies in the absence of normal joint loading and movement after SCI, with a rate of change that is higher than that observed in osteoarthritis (OA). A potential clinical implication is that cartilage thinning after SCI may affect the stress distribution in the joint and render it vulnerable to OA. Future studies should focus on whether specific exercise protocols and rehabilitation programs can prevent cartilage thinning.

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ACCESSION NUMBER: 2003069843 EMBASE

TITLE: Reinforced orbitotemporal lift: Contribution to midface

rejuvenation.

AUTHOR: Reno W.T.

CORPORATE SOURCE: Dr. W.T. Reno, Rua Paissandu 368, Guaratinqueta, Sao Paulo

12 500 121, Brazil

SOURCE: Plastic and Reconstructive Surgery, (2003) Vol. 111, No. 2,

pp. 869-877. .

Refs: 24

ISSN: 0032-1052 CODEN: PRSUAS

COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 009 Surgery

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 20 Feb 2003

Last Updated on STN: 20 Feb 2003

AR The changes in the aging face occur from progressive ptosis of the skin, fat, and muscle, in conjunction with bone absorption and cartilage In the orbital region, hollowness and atrophy. compartmentalization occur. Conventional face lift procedures correct only the skin flaccidity, and superficial musculoaponeurotic system techniques reposition the skin and platysma without repositioning the middle third of the face, creating an artificial jawline. Subperiosteal rhytidectomy disrupts the anatomy of the periorbita, which gives the patient a certain scarecrow aspect. Composite rhytidectomy associated with brow lift and blepharoplasty may offer better results, with improvement in the malar and orbital regions. The reinforced orbitotemporal lift (ROTEL) is a new procedure in a face lift that allows the orbicularis oculi muscle and all the structures connected to it to be elevated and stretched and the orbitotemporal skin to be raised, repositioning these structures and ending orbital compartmentalization. The result is an impressive improvement in the malar-orbitotemporal region, resulting in a natural and youthful appéarance.

reserved on STN

ACCESSION NUMBER: 2000048614 EMBASE

TITLE: Bronchial cartilage atrophy in chronic

bronchitis: observations on chondrolytic processes.

AUTHOR: Tetlow L.C.; Freemont A.J.; Woolley D.E.

CORPORATE SOURCE: Dr. L.C. Tetlow, University Department of Medicine,

Manchester Royal Infirmary, Oxford Road, Manchester M13

9WL, United Kingdom. lynne.c.tetlow@man.ac.uk

SOURCE: Pathobiology, (1999) Vol. 67, No. 4, pp. 196-201. .

Refs: 22

ISSN: 1015-2008 CODEN: PATHEF

COUNTRY: Switzerland DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 005 General Pathology and Pathological Anatomy

> 011 Otorhinolaryngology

015 Chest Diseases, Thoracic Surgery and Tuberculosis

029 Clinical Biochemistry

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 17 Feb 2000

Last Updated on STN: 17 Feb 2000

AB The status of bronchial cartilage degeneration in chronic bronchitis is unclear, and little is known about the chondrolytic mechanisms involved. The potential contributions of various inflammatory cells, chondrocytes and cartilage-degrading enzymes to cartilage atrophy have been examined. Bronchial cartilage specimens were obtained at autopsy from lobar secondary bronchi from chronic bronchitics and age-matched controls; each was examined by light microscopy and immunohistology for the distributions of mast cells, macrophages, eosinophils, collagenase 1, collagenase 3, and degradation products of cartilage collagen. Most bronchitic specimens showed hypertrophic chondrocytes, some of which were immunostained for collagenase 3, and occasionally for collagenase 1. Evidence for collagen degradation products was demonstrated around the lacunae of a proportion of chondrocytes, and both collagenases were also observed in the soft inflammatory tissues in close association with the cartilage surface, together with variable distributions of mast cells and macrophages. observations were generally absent or very much reduced in the control, non-bronchitic specimens. Degenerative changes, atrophy and loss of bronchial cartilage were common features of most chronic bronchitic specimens, this usually being related to intrinsic changes in the chondrocyte phenotype, including proliferative and matrix-degrading properties. Mast cells and macrophages were often observed in close association with the bronchial cartilage, suggesting that inflammatory cells may also contribute to the mechanisms of bronchial cartilage degradation and loss. These observations of bronchial cartilage degeneration were generally lacking in age-matched non-bronchitic control specimens. Copyright (C) 2000 S. Karger AG, Basel.

L1ANSWER 20 OF 33 EMBASE COPYRIGHT (c) 2006 Elsevier B.V. All rights reserved on STN

ACCESSION NUMBER: 1999173382 EMBASE

TITLE: Remobilization does not fully restore immobilization

induced articular cartilage atrophy.

AUTHOR: Haapala J.; Arokoski J.P.A.; Hyttinen M.M.; Lammi M.;

Markku T.; Kovanen V.; Helminen H.J.; Kiviranta I.

CORPORATE SOURCE: Dr. J. Haapala, Harjukatu 48, FIN-15110 Lahti, Finland

SOURCE: Clinical Orthopaedics and Related Research, (1999) No. 362,

pp. 218-229. .

Refs: 39 ISSN: 0009-921X CODEN: CORTBR

COUNTRY: United States

DOCUMENT TYPE: Journal; Conference Article FILE SEGMENT: 033 Orthopedic Surgery

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 3 Jun 1999

Last Updated on STN: 3 Jun 1999

The recovery of articular cartilage from immobilization induced atrophy AB was studied. The right hind limbs of 29-week-old beagle dogs were immobilized for 11 weeks and then remobilized for 50 weeks. Cartilage from the immobilized knee was compared with tissue from age matched control animals. After the immobilization period, uncalcified articular cartilage glycosaminoglycan concentration was reduced by 20% to 23%, the reduction being largest (44%) in the superficial zone. The collagen fibril network showed no significant changes, but the amount of collagen crosslinks was reduced (13.5%) during immobilization. After rembilization, glycosaminoglycan concentration was restored at most sites, except for in the upper parts of uncalcified cartilage in the medial femoral and tibial condyles (9% to 17% less glycosaminoglycans than in controls). The incorporation of 35SO4 was not changed, and remobilization also did not alter the birefringence of collagen fibrils. Remobilization restored the proportion of collagen crosslinks to the control level. The changes induced by joint unloading were reversible at most sites investigated, but full restoration of articular cartilage glycosaminoglycan concentration was not obtained in all sites, even after remobilization for 50 weeks. This suggests that lengthy immobilization of a joint can cause long lasting articular cartilage proteoglycan alterations at the same time as collagen organization remains largely unchanged. Because proteoglycans exert strong influence on the biomechanical properties of cartilage, lengthy immobilization may jeopardize the well being of articular cartilage.

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ACCESSION NUMBER: 95302302 EMBASE

DOCUMENT NUMBER:

1995302302

TITLE:

[Pathology of the locomotory system in ankylosing

spondylitis].

PATHOMORPHOLOGIE DER BEWEGUNGSORGANE BEI DER SPONDYLITIS

ANKYLOSANS.

AUTHOR:

Mohr W.

CORPORATE SOURCE:

Abteilung Pathologie, Universitat Ulm, Albert-Einstein-

Allee 11, D-89081 Ulm, Germany

SOURCE:

Aktuelle Rheumatologie, (1995) Vol. 20, No. 5, pp. 162-170.

ISSN: 0341-051X CODEN: AKRHDB

COUNTRY:

Germany

DOCUMENT TYPE:

Journal; Article

FILE SEGMENT:

005 General Pathology and Pathological Anatomy

031 Arthritis and Rheumatism

LANGUAGE:

German

SUMMARY LANGUAGE:

English; German

ENTRY DATE:

Entered STN: 11 Nov 1995

Last Updated on STN: 11 Nov 1995

AB In the review article pathogenesis and morphology of the different manifestations of ankylosing spondylitis in the locomotory system are described. From the morphological point of view the pathogenesis of the wide spread disease is due to inflammation leading to cartilage and bone destruction. An inflammatory granulation tissue destroys the iliosacral joints, the intervertebral joints and discs and the peripheral joints. The subsequent ossification of the intervertebral discs may lead to the bamboo-spine. The role of a subchondral osteitis in the femoral head and of immobilization on the pathogenesis of cartilage atrophy are briefly discussed.

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ACCESSION NUMBER: 92124282 EMBASE

DOCUMENT NUMBER:

1992124282

TITLE: A mini review: Proteoglycan aggregate profiles in the

Pond-Nuki dog model of osteoarthritis and in canine disuse

atrophy.

AUTHOR: Howel D.S.; Muller F.; Manicourt D.H.

CORPORATE SOURCE:

Department of Medicine, University of Miami, School of Medicine, PO Box 016960, Miami, FL 33101, United States

SOURCE: British Journal of Rheumatology, (1992) Vol. 31, No. 4

SUPPL., pp. 7-11. .

ISSN: 0263-7103 CODEN: BJRHDF

COUNTRY: United Kingdom

DOCUMENT TYPE: Journal; Conference Article

FILE SEGMENT: 031 Arthritis and Rheumatism

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 15 May 1992

Last Updated on STN: 15 May 1992

AB The Pond-Nuki dog model of osteoarthritis has characteristics which seem to mimic the human disease in early stages, particularly with respect to progressive changes in the cartilage matrix. Aggregating proteoglycans were studied using novel extraction and ultracentrifugation methods designed to separate very large macromolecules. With these methods two large peaks of proteoglycan (PG) aggregates (PGA-1 and PGA-2) were separated in preparative amounts and were shown to have unequivocal differences in composition in many respects. The profiles of these peaks have been studied as a function of joint location, topographic site, cartilage layer, presence of cartilage atrophy versus osteoarthritis, as well as treatment of the animals with various agents. Both link protein (essential for forming link-protein stabilized aggregates) and hyaluronate are required to regenerate normal aggregate profiles from the deficient aggregate fractions obtained from osteoarthritic cartilage. Canine proteoglycan link-stabilized aggregates (PGA-2) are confined to the middle and deep zone of cartilage. We believe that their reduction or elimination in the Pond-Nuki model results from a disturbance or loss of function link protein (and hyaluronate), there by weakening the middle and deep cartilage layers.

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90263666 EMBASE ACCESSION NUMBER:

DOCUMENT NUMBER: 1990263666

TITLE: Pathophysiology of chronic obstructive pulmonary disease.

AUTHOR: Thurlbeck W.M.

CORPORATE SOURCE: Department of Pathology, University of British Columbia,

2211 Westbrook Mall, Vancouver, BC V6T 1W5, Canada Clinics in Chest Medicine, (1990) Vol. 11, No. 3, pp.

389-403.

SOURCE:

ISSN: 0272-5231 CODEN: CCHMDA

COUNTRY: United States

DOCUMENT TYPE: Journal; General Review

FILE SEGMENT: 005 General Pathology and Pathological Anatomy

> 006 Internal Medicine

015 Chest Diseases, Thoracic Surgery and Tuberculosis

LANGUAGE: English SUMMARY LANGUAGE: English

ENTRY DATE: Entered STN: 13 Dec 1991

Last Updated on STN: 13 Dec 1991

AB Chronic airflow obstruction (CAO) is a syndrome that is produced by a variety of lesions which may occur in bronchi (large airways), bronchioles (small airways), and lung parenchyma (gas exchanging lung). These lesions frequently occur together in various combinations because of a common etiologic agent, tobacco smoke. Occasionally, one lesion or another may play a dominant role. The major disease of the large airways is chronic bronchitis, or chronic sputum production, and it is defined clinically. Its morphologic counterpart is mucous gland enlargement. Mucous gland enlargement is poorly related to CAO. Other lesions of the large airways

- inflammation, smooth muscle hyperplasia, cartilage atrophy, and bronchial wall thickening - have also been described, but their functional consequences are uncertain. Bronchiolar lesions are well recognized in CAO, but their relative importance may differ in patients with mild CAO, compared to patients with severe CAO. In mild CAO, inflammation is a very important lesion, and its probable consequences - narrowing, fibrosis, and goblet cell metaplasia - have all been found to be important. In severe CAO, inflammation and fibrosis do not appear to be important, but goblet cell metaplasia, bronchiolar tortuosity, and narrowing do. Emphysema is a subset of airspace enlargement. Emphysema is defined anatomically and is the most important component of severe CAO. Several forms of emphysema can be recognized morphologically and may have specific clinical associations. However, in the usual patient with severe CAO, it is the severity, rather than the type, of emphysema, that is most significant. The diagnosis of emphysema depends on a combined approach. Significant factors include the clinical history (age, sex, smoking, chronic bronchitis, dyspnea), radiologic evidence of overinflation, and diminished diffusing capacity for carbon monoxide.

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reserved on STN ACCESSION NUMBER:

90040671 EMBASE

DOCUMENT NUMBER:

1990040671

TITLE:

Pathology of chronic airflow obstruction.

AUTHOR:

Thurlbeck W.M.

CORPORATE SOURCE:

University of British Columbia, Faculty of Medicine,

SOURCE:

Vancouver, BC, Canada Chest, (1990) Vol. 97, No. 2 SUPPL., pp. 6S-10S. .

ISSN: 0012-3692 CODEN: CHETBF

COUNTRY:

United States

DOCUMENT TYPE:

Journal; Conference Article

FILE SEGMENT:

005 General Pathology and Pathological Anatomy

015 Chest Diseases, Thoracic Surgery and Tuberculosis

LANGUAGE:

English

SUMMARY LANGUAGE: ENTRY DATE:

English Entered STN: 13 Dec 1991

Last Updated on STN: 13 Dec 1991

Classification of chronic airflow obstruction may be based on the site of the obstructing lesions. It is seldom that only one type of lesion is present, but one may often dominate. In chronic bronchitis, the major disease of large airways, chronic mucus hypersecretion, is reflected by an increase in size of bronchial mucous glands. This may be a factor in airway narrowing, especially with coexisting edema of the airway wall. Excess intralumenal mucus compounds the obstruction. Increased airways reactivity is present in 15 to 70 percent of patients with chronic airflow obstruction. Increased airway muscle and cartilage atrophy are features of chronic bronchitis, but the association of increased muscle with increased airway reactivity is poor. Inflammation of the small airways (bronchiolitis) is a significant complication for cigarette smokers and is an important cause of mild chronic airflow obstruction. Goblet cell metaplasia is a reflection of chronic small airways inflammation and, together with intralumenal mucus, is an important feature. Permanent narrowing of the small airways presumably results from inflammation with consequent fibrosis, while functional narrowing results from release of mediators of inflammation. Increased muscle mass is present in some cases. Distortion and irregularity of small airways related to emphysema are major factors in severe obstruction. Lesser degrees of emphysema may be associated with a diminished number of alveolar attachments and mild chronic airflow obstruction. Emphysema, the dominant lesion in patients with severe chronic airflow obstruction, results from parenchymal lesions. Centrilobular emphysema, in which the respiratory bronchioles are selectively or dominantly involved, is the most common form. Familial al-antiprotease deficiency is the classic example of panacinar

emphysema. When severe, this condition is dominantly a lower zonal disease.

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ACCESSION NUMBER: 89272275 EMBASE

DOCUMENT NUMBER:

1989272275

TITLE:

Cartilage matrix glycoprotein is present in serum in

experimental canine osteoarthritis.

AUTHOR:

Fife R.S.; Brandt K.D.

CORPORATE SOURCE:

Rheumatology Division, Department of Medicine, Indiana University School of Medicine, Indianapolis, IN 46202,

United States

SOURCE:

Journal of Clinical Investigation, (1989) Vol. 84, No. 5,

pp. 1432-1439.

ISSN: 0021-9738 CODEN: JCINAO

COUNTRY:

United States

DOCUMENT TYPE:

Journal

FILE SEGMENT:

029 Clinical Biochemistry 031 Arthritis and Rheumatism

LANGUAGE: SUMMARY LANGUAGE: English English

ENTRY DATE:

Entered STN: 12 Dec 1991

Last Updated on STN: 12 Dec 1991

AB We have described previously a disulfide-bonded 550,000-D cartilage matrix glycoprotein (CMGP), which is found in normal hyaline cartilage, fibrocartilage, and the vitreous of the eye, and consists of subunits with apparent molecular weights of 130,000 in 4% gels (116,000 in 9% gels). In osteoarthritic cartilage from dogs subjected to transection of the anterior cruciate ligament (ACL), CMGP is cleaved to major immunoreactive fragments with apparent molecular weights of 65,000 and 75,000 after reduction with 2-mercaptoethanol. In the present study, using immunolocation analysis, a monoclonal antibody to CMGP did not react with serum from 8 of 12 dogs before ACL transection but did react with serum from seven of these animals 4 wk after surgery and with serum from 10 dogs at sacrifice, 8-14 wk after ACL transection. Serum from four dogs reacted with the monoclonal antibody before ACL transection. Serum from two dogs was negative at all time points. Immunolocation studies using a polyclonal antiserum to CMGP were performed in seven of these dogs and produced results identical with the monoclonal antibody in four dogs. contrast, analysis of serial serum samples from these three dogs with cartilage atrophy revealed no evidence of CMGP at any These data suggest that CMGP may be a serum marker for time point. osteoarthritis in this canine model.

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ACCESSION NUMBER:

86101093 EMBASE

DOCUMENT NUMBER:

1986101093

TITLE:

Rheumatoid articular pannus. Histogenesis and the mechanism

of articular cartilage destruction by pannus.

AUTHOR:

Wierzchowska E.; Maldyk E.

CORPORATE SOURCE:

Zakladu Anatomii Patologicznej Instytutu Reumatologicznego,

Warszawa, Poland

SOURCE:

Patologia Polska, (1985) Vol. 36, No. 2, pp. 178-186. .

CODEN: PAPOAC

COUNTRY:

Poland Journal

DOCUMENT TYPE:

031 Arthritis and Rheumatism

FILE SEGMENT: 005

General Pathology and Pathological Anatomy

LANGUAGE:

Polish

SUMMARY LANGUAGE:

English; Russian

ENTRY DATE:

Entered STN: 10 Dec 1991 Last Updated on STN: 10 Dec 1991

AB Atricular pannus in 40 women and 10 men with classic or definite rheumatoid arthritis was examined histopathologically and histochemically. The results of the examinations show that there are two types of pannus: inactive and active one. Inactive pannus is a poorly vascularized coat connective tissue coering the surface of articular cartilage. Active pannus has a connective tissue of loose structure and its cells penetrate deeply into the cartilage in a similar manner as neoplastic cells. In both cases destruction and articular cartilage atrophy are the endpoints.

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ACCESSION NUMBER: 84207067 EMBASE

DOCUMENT NUMBER: 1984207067

TITLE: Synovectomy as treatment for purulent joint infection.

AUTHOR: Tscherne H.; Giebel G.; Muhr G.; Howell Ch.

CORPORATE SOURCE: Unfallchirurgische Klinik der Medizinischen Hochschule,

D-3000 Hannover 61, Germany

SOURCE: Archives of Orthopaedic and Traumatic Surgery, (1984) Vol.

103, No. 3, pp. 162-164. .

CODEN: AOUNAZ

COUNTRY: Germany DOCUMENT TYPE: Journal

FILE SEGMENT: 033 Orthopedic Surgery

031 Arthritis and Rheumatism

004 Microbiology

LANGUAGE: English SUMMARY LANGUAGE: German

ENTRY DATE: Entered STN: 10 Dec 1991

Last Updated on STN: 10 Dec 1991

AB Conventional treatment of pyogenic knee joint infections leads to unsatisfactory results. Through early synovectomy, before cartilage damage and osteoarthritis appear, the infected focus can be 'excised'. Functional after-treatment avoids cartilage atrophy,

wound adhesions, and muscle weakness. The excellent results after 26 knee joint infections confirm this.

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ACCESSION NUMBER: 84175147 EMBASE

DOCUMENT NUMBER: 1984175147

TITLE: Effects of salicylates and other nonsteroidal anti-inflammatory drugs on articular cartilage.

AUTHOR: Brandt K.D.; Palmoski M.J.

CORPORATE SOURCE: Rheumatology Division, Indiana University Medical Center,

Indianapolis, IN 46223, United States

SOURCE: American Journal of Medicine, (1984) Vol. 77, No. 1 A, pp.

65-69. .

COUNTRY: CODEN: AJMEAZ United States

DOCUMENT TYPE: Journal

FILE SEGMENT: 037 Drug Literature Index

030 Pharmacology

031 Arthritis and Rheumatism

033 Orthopedic Surgery

LANGUAGE: English

ENTRY DATE: Entered STN: 10 Dec 1991

Last Updated on STN: 10 Dec 1991

AB According to in vivo experimental data, salicylates and several other nonsteroidal anti-inflammatory agents suppress proteoglycan biosynthesis in normal and degenerating articular cartilage. Therapeutic levels of aspirin in vivo had a similar adverse effect on degenerating cartilage, as noted in two canine models of osteoarthritis and cartilage atrophy. Because the effective daily antirheumatic dose of nonsteroidal anti-inflammatory drugs is lower than that of salicylates, these drugs may have less negative effects on degenerating articular

cartilage. However, clinical significance cannot be extrapolated from these experimental data.

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ACCESSION NUMBER: 82053924 EMBASE

DOCUMENT NUMBER:

1982053924

TITLE:

Articular cartilage atrophy in lower

limb amputees.

AUTHOR:

Benichou C.; Wirotius J.M.

CORPORATE SOURCE:

Cent. Hosp. St Cloud, 92210 St Cloud, France

SOURCE:

Arthritis and Rheumatism, (1982) Vol. 25, No. 1, pp. 80-82.

CODEN: ARHEAW

COUNTRY:

United States

DOCUMENT TYPE:

Journal

FILE SEGMENT:

031 Arthritis and Rheumatism

033

Orthopedic Surgery 005 General Pathology and Pathological Anatomy

LANGUAGE:

English

ENTRY DATE:

Entered STN: 9 Dec 1991

Last Updated on STN: 9 Dec 1991

AB A retrospective and radiologic surveys of the hips of 53 above knee amputees showed that none of these hips was normal. Osteoporosis was present in all subjects, and cartilage thickness was reduced in 27 cases. This reduced thickness was inversely correlated with stump length, since it occurred in 11 of 13 upper third amputees, but in none of 10 lower third amputees. The mechanisms of cartilage atrophy are discussed.

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ACCESSION NUMBER:

82005334 EMBASE

DOCUMENT NUMBER:

1982005334

TITLE:

Running inhibits the reversal of atrophic changes in canine

knee cartilage after removal of a leg cast.

AUTHOR:

Palmoski M.J.; Brandt K.D.

CORPORATE SOURCE:

Rheumatol. Div., Indiana Univ. Sch. Med., Indianapolis, IN

46223, United States

SOURCE:

Arthritis and Rheumatism, (1981) Vol. 24, No. 11, pp.

1329-1337. . CODEN: ARHEAW United States

COUNTRY: DOCUMENT TYPE:

Journal

FILE SEGMENT:

031 Arthritis and Rheumatism

LANGUAGE:

English

ENTRY DATE:

Entered STN: 9 Dec 1991

Last Updated on STN: 9 Dec 1991

The effect of vigorous exercise on the reversibility of canine knee cartilage atrophy produced by immobilization of the leg was studied. In comparison to cartilage from the contralateral control knees, cartilage from knees which had been immobilized in a cast for 6 weeks showed an increase in water content and decreases in thickness, Safranin O staining of the matrix, uronic acid content, and net proteoglycan synthesis. In addition, the ability of both newly synthesized (35S) and total tissue proteoglycans to interact with hyaluronic acid to form aggregates was diminished; this was apparently due to an abnormality in the hyaluronate-binding region of the core proteins. If the casts were removed and the animals were then allowed to ambulate ad libitum for 3 weeks, all of these changes were reversed. However, knee cartilage from 3 dogs which had been run daily on a treadmill (6 miles/day) for 3 weeks after removal of the casts exhibited continuing decreases in thickness, Safranin O staining, and uronic acid content (mean 31%), even though net proteoglycan synthesis was increased (mean 16%) in comparison to that in control cartilage from the contralateral

(nonimmobilized) knee. Furthermore, the abnormality in both 35S- and total tissue proteoglycans which precluded their interaction with high molecular weight hyaluronic acid persisted. In this respect, the proteoglycans were indistinguishable from those obtained from knee cartilage immediately following 6 weeks in a cast.

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ACCESSION NUMBER: 81232043 EMBASE

DOCUMENT NUMBER:

1981232043

TITLE:

Effect of joint disuse and subsequent exercise on proteoglycan mtabolism and aggregation in articular

cartilage.

AUTHOR:

Palmoski M.; Brandt K.

CORPORATE SOURCE: SOURCE:

Indiana Univ. Sch. Med., Indianapolis, IN, United States Seminars in Arthritis and Rheumatism, (1981) Vol. 11, No. 1

Suppl. 1, pp. 30-31. .

CODEN: SAHRBF

COUNTRY:

United States

DOCUMENT TYPE:

Journal

FILE SEGMENT:

031 Arthritis and Rheumatism

019 Rehabilitation and Physical Medicine

029 Clinical Biochemistry

LANGUAGE:

English

ENTRY DATE:

Entered STN: 9 Dec 1991

Last Updated on STN: 9 Dec 1991

AB We have described changes in knee cartilage after amputation of the ipsilateral paw identical to changes produced by immobilization. Notably, the cartilage degeneration in the paw transection model developed in the presence of a normal arc of knee movement, strongly suggesting that the changes arising with immobilizaton were not due simply to a lack of joint motion but to reduction in the loading of the cartilage, which results from contraction of the muscles that span the joint and stabilize the limb instance. The objective of the present study was to determine whether the reversibility of the cartilage atrophy induced by immobilization might be affected by vigorous but physiologic usage of the recently constrained joint.

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ACCESSION NUMBER:

81190509 EMBASE

DOCUMENT NUMBER:

1981190509

TITLE:

Cartilage atrophy following spinal cord

damage.

AUTHOR:

Anderson J.; Breidahl P.

CORPORATE SOURCE:

Dept. Diagn. Radiol., Roy. Perth Hosp., Perth, WA,

Australia

SOURCE:

Australasian Radiology, (1981) Vol. 25, No. 1, pp. 98-103.

CODEN: AURDAW

COUNTRY:

Australia Journal

DOCUMENT TYPE:

Radiology 014

FILE SEGMENT:

033

Orthopedic Surgery

800 Neurology and Neurosurgery

LANGUAGE:

English

ENTRY DATE:

Entered STN: 9 Dec 1991

Last Updated on STN: 9 Dec 1991

AΒ A retrospective analysis of hip joint space measurement of patients who had previously sustained spinal cord damage was undertaken. This paper confirms previous findings that patients with flaccid paralysis in lower limbs develop hip joint space narrowing. It has been found that this phenomenon is far more frequent than previously reported and can be seen in a significant proportion of patients with lower limb spasticity.

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75065794 EMBASE ACCESSION NUMBER:

DOCUMENT NUMBER:

1975065794

TITLE:

Cartilage atrophy.

AUTHOR:

Pool Jr W.H.

CORPORATE SOURCE:

Dept. Radiol., Med. Coll. Georgia, Augusta, Ga., United

States

SOURCE:

Radiology, (1974) Vol. 112, No. 1, pp. 47-50. .

CODEN: RADLAX

DOCUMENT TYPE:

Journal

English

FILE SEGMENT:

014 Radiology

031

Arthritis and Rheumatism

033 Orthopedic Surgery

LANGUAGE:

Two hundred cases of flaccid paralysis of the lower extremities were reviewed, and in 25 the cartilaginous space of the hip joint was found to be narrowed by at least 50%. It is postulated that the cartilage

reduction is the result of atrophy following an altered nutrition associated with lack of stress and a decrease in the production of

synovial fluid.

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